Case Report

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Ablation of ventricular tachycardia by direct left ventricle puncture through a minithoracotomy after double valve replacement: a case report and literature review

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Abstract

We herein describe a 33-year-old woman with a mechanical aortic and mitral valve who developed repetitive monomorphic ventricular tachycardia with unstable hemodynamics. Catheter ablation by direct puncture at the left ventricular apex through a minithoracotomy successfully terminated the ventricular tachycardia, which had originated from the apical-septal endocardium in the left ventricle, despite the hindrance to routine access. No procedure-related complications or recurrence of the clinical ventricular tachycardia developed during a 66-month follow-up, demonstrating that endocardial ablation through direct cardiac cavity puncture can be considered in select cases.

Keywords

Ventricular tachycardia, catheter ablation, mechanical prosthetic valve, minithoracotomy, rheumatic heart disease, cardiac puncture

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Introduction

Ventricular arrhythmia (VA) is common in patients with rheumatic heart disease (RHD), which can cause deterioration in cardiac function. Catheter ablation can reduce the VA burden and improve

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patients' symptoms and cardiac function. However, catheter ablation is challenging to perform in patients with mechanical prosthetic aortic and mitral valves because conventional access into the left ventricle (LV), either through the atrial trans-septal approach or retrograde aortic approach, may be hindered by the prosthetic valves. We herein present a case of successful catheter ablation for nearly incessant ventricular tachycardia (VT), which was achieved by direct LV puncture through a minithoracotomy. We also present a review of the available therapy choices for VA originating from the LV after double valve replacement.

Case report

A 33-year-old woman with advanced RHD was admitted with the chief complaint of recurrent palpitation and worsening dyspnea. The patient had undergone metal mechanical mitral and aortic valve replacement as well as tricuspid valve repair 1.5 years before this admission. Despite the successful valve replacement surgery, the patient still experienced severe symptoms due to frequent palpitation and deterioration of heart function. The electrocardiogram (ECG) on admission showed repetitive monomorphic VT at a rate of 90 to 100 beats/minute, which was poorly tolerated and caused the blood pressure to drop during tachycardia onset. The ECG morphology of the clinical VT indicated an apical origin, with a left bundle branch block pattern and leftward axis (Figure 1). An echocardiogram revealed global LV wall hypokinesis with a left ventricular ejection fraction (LVEF) of 23.9% and a dramatically dilated right ventricle. The prosthetic valves were functioning normally. Laboratory test results were significant for a B-type natriuretic peptide concentration of 5773 pg/mL and slightly elevated inflammatory markers (erythrocyte sedimentation rate, 20 mm/hour; high-sensitivity C-reactive protein concentration, 19.8 mg/L). Drug therapy with amiodarone and sotalol was not effective.

An implantable cardioverter defibrillator (ICD) was indicated because of the nearly incessant VT and decreased cardiac function. However, the patient refused ICD implantation. Because the clinical VT was monomorphic, catheter ablation was considered. However, with the mechanical prosthetic mitral and aortic valves, the routine ablation approach via retrograde aortic access or atrial trans-septal access into the LV were not feasible. To reach the LV cavity, direct LV puncture by a minithoracotomy was proposed to create artificial access for the subsequent catheter ablation. The patient agreed to this procedure and provided written informed consent.

The procedure was performed in a hybrid operating room. The patient still exhibited incessant VT during the procedure. Local activation mapping with a three-dimensional electroanatomical mapping system (EnSite 3 Cardiac Mapping System; Abbott Laboratories, Chicago, IL, USA) was performed in the right ventricle using a mapping and ablation catheter (Cool Flex; St. Jude Medical, Little Canada, MN, USA), but this procedure did not reveal activation earlier than the QRS complex on the surface ECG. Thus, after administration of general anesthesia, an 8-cm incision was created along the sixth intercostal space. The pericardial adhesion from the previous surgery was dissected, and the LV apex was clearly exposed through a left anterolateral minithoracotomy (Figure 2). Unfractionated heparin was given to maintain an activated clotting time (ACT) of 250 to 300 seconds. The same catheter was advanced into the LV cavity though a 7-French sheath by direct puncture at the LV apex on the free wall side, and activation mapping was

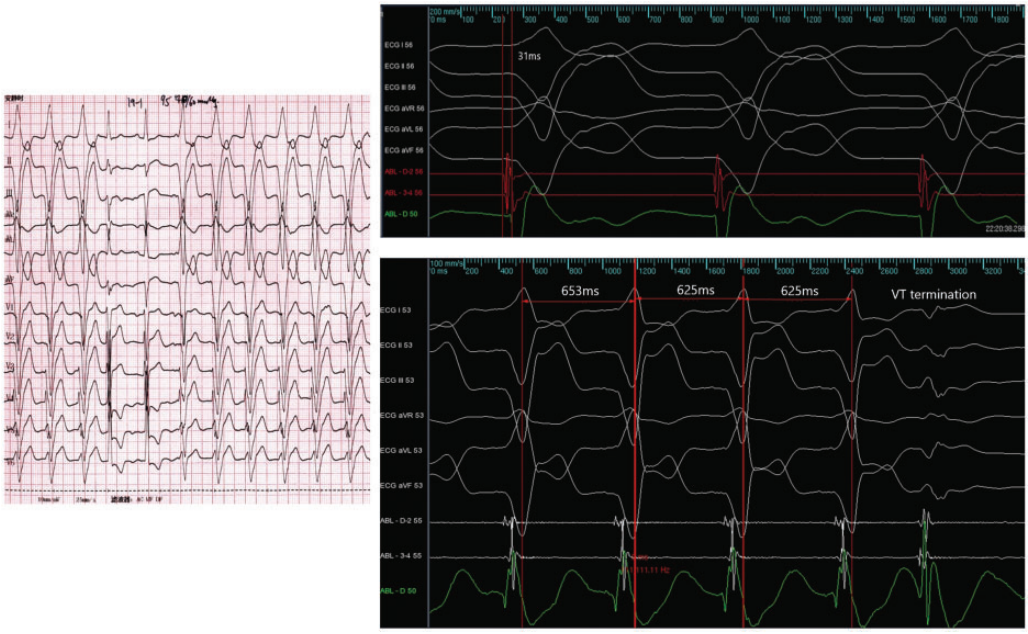

Figure 1. (a) Twelve-lead rhythm strip of ventricular tachycardia (VT) showing left bundle branch block morphology with left axis deviation. (b) Intracardiac recordings from the endocardial left ventricle. The earliest activation occurred 31 ms before the onset of the QRS on the surface electrocardiogram. (c) Termination of the VT during ablation at the site shown in Figure 1(b). The clinical VT terminated after a premature ventricular contraction. No spontaneous VT recurred, and the VT remained noninducible with repetitive programmed electrical stimulation after administration of intravenous isoproterenol.

conducted. Because the puncture hole was small and stable hemodynamics could be maintained, the patient was not placed on extracorporeal circulation. Fluoroscopy was used to visualize the location of the catheter to avoid mapping too close to the artificial valves. Activation mapping at the septal apical LV endocardium showed that the earliest activation preceded the onset of the QRS complex on the surface ECG for 31 ms (Figure 3). Unipolar mapping revealed a QS morphology with a sharp notch in the initial part. Ablation with an energy of 35 to 45 W at 42°C was delivered at this site. The initial ablation repressed the frequency of the VT episodes; however, the refractoriness of the VT implicated that the proarrhythmic substrate of the VT was deep within the ventricular myocardium. Repeated ablation with an enhanced power of 55 W for 40 seconds was applied, and the clinical VT terminated after a premature ventricular contraction. No spontaneous VT recurred, and the VT remained noninducible with repetitive programmed electrical stimulation after administration of intravenous isoproterenol. The sheath and catheter were withdrawn, and hemostasis was achieved by tying purse-string sutures to close the access site. The total procedure time was 142 minutes, and the fluoroscopy time was 13 minutes.

The patient was transferred to the surgical intensive care unit after the procedure and recovered well. Warfarin was started 2 days after the ablation procedure. Cefoperazone–sulbactam and moxifloxacin were administered for 1 week after the

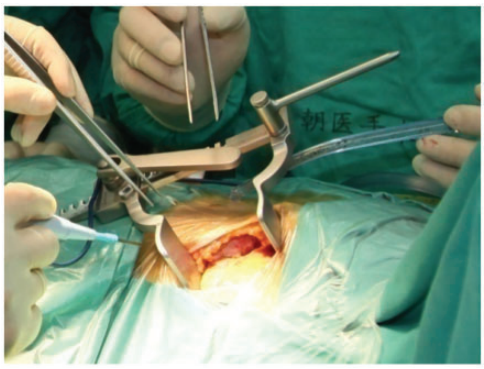

Figure 2. Surgical access through the apex.

procedure because the patient developed a lung infection. No clinical arrhythmia occurred after the procedure; therefore, no antiarrhythmic drug was prescribed. The patient's cardiac function improved with an increase in the LVEF from 23% before the procedure to 31% within 1 month. During a 66-month follow-up, no palpitation occurred and annual dynamic ECGs showed normal sinus rhythm without VA recurrence. An echocardiogram at 66 months revealed an LVEF of 61% with normal prosthetic valve function.

The Ethics Committee of Beijing Chaoyang Hospital approved submission and publication of this case report.

Discussion

The vast majority of VAs in patients with RHD originate from the endocardium.[1] According to the current guideline, VT in patients with structural heart disease with unstable hemodynamics is a class I indication for ICD implantation. However, incessant VT leads to repeated discharge of the ICD, causing pain and anxiety to the patient, as well as premature energy depletion of the ICD, which may impact patients' acceptance of this treatment. Because antiarrhythmic drugs were ineffective in the present case, catheter ablation was the only treatment choice.

The main challenge when performing VT radiofrequency ablation in patients with mechanical prosthetic valves lies in the possibility of prosthetic valve dysfunction and damage caused by the catheter. Because the catheter must pass through the mechanical mitral valve to enter the LV, it may cause valvular insufficiency and significant regurgitation. Repeated catheter manipulation may lead to mechanical valvular damage, which may result in cardiac function deterioration, acute hemodynamic instability, or even catheter entrapment in the prosthetic valves, which may require surgical intervention. No standard management protocol has been established for this clinical problem. Previously reported cases of VT complicated by mechanical aortic and mitral valves are summarized in Table 1.

With the support of a left ventricular assist device,[2] Herweg et al.[2] introduced a catheter into the LV cavity through a mechanical mitral valve in a critically ill patient. However, a left ventricular assist device is not routinely available in patients with VT undergoing catheter ablation to maintain stable hemodynamics. Epicardial ablation in patients with double-valve replacement has been reported in a series of cases.[3–7] Although proven effective for VT originating from the epicardium, its application may be restricted by its lower efficacy in treating endocardial VT as well as severe adhesion of the pericardium caused by previous cardiac surgery. Ablation of the epicardial VT through the great cardiac vein[6] is also reportedly effective, but its applicability might be limited by an apical endocardium origin, as in our case. Percutaneous trans-interventricular septum approaches were adopted in several pilot case reports.[8,9] However, because our patient had a history of tricuspid valvuloplasty, the passage of the catheter may

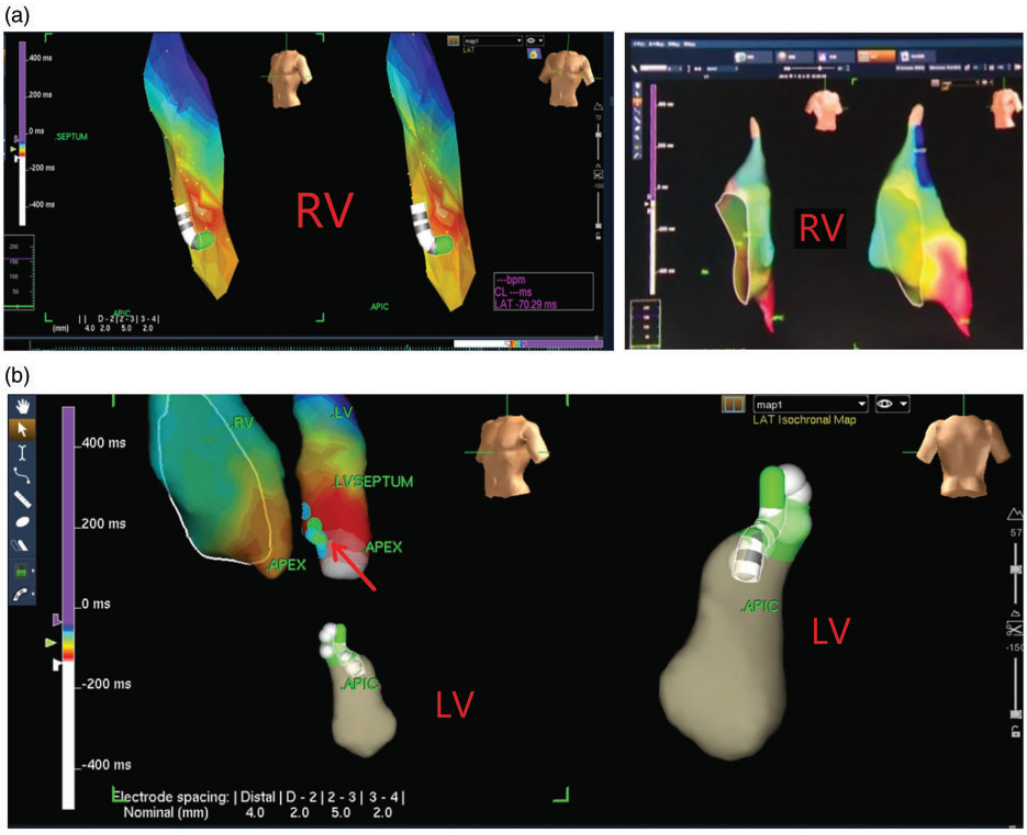

Figure 3. (a) Activation mapping in the right ventricle (RV). The earliest activation site in the RV was near the apical septum. However, activation mapping in the RV did not reveal activation earlier than the QRS complex on the surface electrocardiogram. (b) Activation mapping in the left ventricle at the septal-apical endocardium revealed the earliest activation, which preceded the onset of the QRS complex on the surface electrocardiogram for 31 ms. Unipolar mapping revealed a QS morphology with a sharp notch in the initial part (corresponding to Figure 1). The right panel B shows the mapping process.

affect the function of the tricuspid valve. Besides radiofrequency ablation, Baldinger et al.[10] presented a case of VT termination by transcoronary ethanol ablation. Notably, lesions treated by transcoronary ethanol ablation are difficult to control; thus, such an option must be meticulously evaluated before application.

LV puncture through a minithoracotomy provides the most direct approach to the LV cavity with relatively fewer limitations compared with the transvascular approach. The LV can be accessed either by blind percutaneous puncture[11,12] or under direct vision with the assistance of a minithoracotomy. However, methods without clear exposure of the puncture site carry a high risk of complications, such as pneumothorax, hemothorax, or even cardiac tamponade due to injury of the coronary artery; another potential complication is persistent apical leakage after sheath withdrawal, especially in patients undergoing systemic anticoagulation.[13] An anterior-

Table 1. Demographic, clinical, and substrate location features of the reviewed cases of VT ablation in patients with mitral and aortic prosthetic valves.

Authors	Year of publication	Patient age (years)	Sex	Pathogenesis	Past cardiac surgery	VA substrate	LV access	Complication	Follow-up
Hsieh et al.[13]	2010	65	Male	RHD, CAD, DCM, PAF	MVR, AVR, ICD	Anterobasal LV, endocardium	Percutaneous puncture through the intercostal space	Large left hemothorax	4 months of antitachycardia pacing terminated one VT episode
		66	Male	RHD, HF, AF, AVB	MVR, AVR, ICD	Inferior-apical LV, endocardium	Left minithoracotomy	None	2 months arrhythmia-free
Reents et al.[14]	2014	48	Male	IE	MVR, AVR, CPT-D	Inferior LV, endocardium	Left anterolateral minithoracotomy at the fifth intercostal space	None	6 months arrhythmia-free
Menon et al.[15]	2017	63	Male	Congenital bicuspid aortic valve infection	MVR, AVR, CABG, CRT-D	Inferior-apical LV, endocardium	Anterolateral thoracotomy and LV lateral wall puncture	None	14 months arrhythmia-free
Santangeli et al.[9]	2017	53	Female	RHD	MVR, AVR, ICD	LV apical aneurysm, endocardium	Percutaneous trans-interventricular septum	None	4 months arrhythmia-free
Vaseghi et al.[8]	2013	35	Male	IE	MVR, AVR, ICD	Exit at mid-inferior wall of the LV, endocardium	Percutaneous trans-interventricular septum	None	Not reported
Herweg et al.[2]	2010	72	Male	RHD, HF	MVR, AVR, ICD, and LVAD	Basal and mid-portions of the LV, endocardium	Trans-septal catheterization passing the mechanical valve	None	10 weeks shock-free after a second ablation procedure 10 weeks after the first ablation procedure
Anh et al.[3]	2007	41	Female	RHD, CAD, IC	MVR, AVR	LV inferior base, epicardium	Subxiphoid incision	Mild pericarditis	5 months arrhythmia-free
Najjar et al.[6]	2007	60	Female	N/A	MVR, AVR	Distal aspect of GCV, epicardium	Mapping and ablation trough GCV	None	9 months arrhythmia-free
Maury et al.[7]	2009	62	Female	RHD	MVR, AVR	Apical and superior in LV free wall, epicardium	Left anterolateral thoracotomy at the fifth intercostal space	None	Sporadic isolated VT episodes during 9-month follow-up

(continued)

Table 1. Continued

Authors	Year of publication	Patient age (years)	Sex	Pathogenesis	Past cardiac surgery	VA substrate	LV access	Complication	Follow-up
Soejima et al.[4]	2015	27	Male	RHD	MVR, AVR	Lateral apical LV, epicardium	Open-heart surgical cryoablation	None	18 months arrhythmia-free
		35	Male	RHD	MVR, AVR	LV apex and inferior wall, epicardium	Surgical epicardial access	None	2 months arrhythmia-free
		40	Male	RHD	MVR, AVR	LV apical, epicardium	Surgical epicardial access	None	18 months arrhythmia-free
		25	Male	RHD	MVR, AVR	LV anterior aneurysm, epicardium	Open-heart aneurysmectomy and cryoablation	None	Not reported
		21	Male	RHD	MVR, AVR	LV inferior-lateral wall, epicardium	Surgical epicardial access	None	Died within 1 week
		29	Male	RHD, IE	MVR, AVR	Exit at distal portion of the middle cardiac vein, epicardium	Open-heart surgical cryoablation	None	Not reported
Vurgun et al.[5]	2018	57	Male	HCM, IE	MVR, AVR, PM	LV mid-lateral segment, epicardium	Apical puncture	None	15 months arrhythmia-free
Baldinger et al.[10]	2015	52	Male	RHD, CAD	MVR, AVR, ICD	Lateral LV scar, endocardium	Balloon occlusion and ethanol injection into the distal branches of a marginal branch	None	No further VT, died 2 months later

AF, atrial fibrillation; AVB, atrioventricular heart block; AVR, aortic valve replacement; CABG, coronary artery bypass graft; CAD, coronary artery disease; DCM, dilated cardiomyopathy; GCV, great cardiac vein; HCM, hypertrophic cardiomyopathy; HF, heart failure; IC, ischemic cardiomyopathy; ICD, implantable cardioverter defibrillator; IE, infectious endocarditis; LV, left ventricle; LVAD, left ventricular assist device; MVR, mitral valve replacement; PM, pacemaker; RHD, rheumatic heart disease; TVR, tricuspid valve replacement; VT, ventricular tachycardia.

lateral minithoracotomy can clearly expose the LV apex, thus allowing direct visualization of the puncture site and placement of a purse-string suture with pledgets to guarantee effective hemostasis.[13–15] Considering the lower complication rate compared with blind puncture, a minithoracotomy should be the first choice, especially in patients with LV systolic dysfunction whose depressed myocardial contraction cannot effectively promote closure of the puncture hole. The safety and efficacy of this method have been proven by sporadic reports in patients with ICDs, and our case demonstrates that when the application of an ICD is limited, catheter ablation by direct LV access through a minithoracotomy remains a plausible therapeutic choice.

The main limitation of our case lies in the lack of further investigation regarding the mechanism underlying the VT in this patient. Although we assumed that the proarrhythmic substrate was associated with the chronic rheumatic carditis, relevant imaging examinations and pathological evidence are needed to corroborate our hypothesis.

Conclusion

VT ablation by direct transthoracic access into the LV endocardium can be considered in select patients after double prosthetic valve replacement wen conventional transvascular approaches are inappropriate.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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